Wilson/Fenton/Tompkins - Wildlife Disease Ecology: Lay summaries and Abstracts

Chapter 1 Pollinator diseases: The *Bombus-Crithidia* system

Paul Schmid-Hempel, Lena Wilfert & Regula Schmid-Hempel

Lay summary

Bumblebees are social insects that form small colonies, and are commonly infected by single-celled gut parasites of the genus *Crithidia*. Successful infection depends on a suitable strain, i.e. a genotype, of the parasite encountering a susceptible variant (colony) of the host. In any one field population, the genotypic diversity of the parasite is very large. This diversity is known to be generated by the frequent occurrence of genetic exchange between co-infecting parasite strains through recombination during sexual reproduction. Hosts defend themselves, among other things, by producing very effective "natural antibiotics", anti-microbial peptides, that work together to control or eliminate the infection.

Abstract

Several species of *Crithidia* (Trypanosomatida) are gut parasites of bumble bees (*Bombus* spp.). Their main effect is to castrate the queens emerging from hibernation in spring. The parasites are common in natural populations and spread among colonies and host species via the shared use of flowers. Infection prevalence varies on a small geographic scale and over years, but with no clear pattern that can explain the differences. Genetic analyses of the best-investigated species, *C. bombi*, shows that mixed-genotype infections of single host individuals are common in the field, and associated with a relatively high rate of sexual reproduction inside the host. In fact, parasite populations in the field, and also within a host colony or individual are genotypically highly diverse, with up to around 30 concurrent infections, such that the same genotype is almost never found more than once among a sample of infected bees. A hallmark of this system is that a given parasite genotype infects only a certain range of host genotypic backgrounds (i.e. colonies), and this range varies among parasite genotypes. In turn, hosts are also susceptible only to a limited range of parasite genotypes. A major defence that *Bombus* uses against *Crithidia* is by deployment of anti-microbial peptides (AMPs), which have been characterized with the release of the *Bombus* genome. Experiments show that AMPs have synergistic effects. Queen mothers can furthermore protect their offspring, the workers in the colony, by trans-generational immune priming.

Chapter 2 **Genetic diversity and disease spread: epidemiological models and empirical studies of a snail-trematode system**

Amanda K. Gibson & Curtis M. Lively

Lay summary

Does genetic diversity of a host population limit the spread of an infectious disease? Here, we address this question by drawing upon theoretical models from different disciplines. We then connect the models to patterns uncovered in a natural host-parasite system, the freshwater snail *Potamopyrgus antipodarum* and its sterilising trematode parasite *Microphallus*. Both models and field data suggest that genetic diversity in host resistance can limit the spread of a parasite. Expanding upon this basic idea, we show that disease spread can increase if parasites adapt to infect common host genotypes. We discuss the implications of these results for the maintenance of sexual reproduction, a costly strategy that is nonetheless widespread in nature. We emphasize that the results we summarize here emerge only when we consider both epidemiology and genetics.

Abstract

For simplicity's sake, standard population genetic models of host-parasite coevolution often exclude ecological and epidemiological detail. In particular, they assume that each host is exposed to a single infectious propagule, regardless of the prevalence of the parasite. On the other hand, standard epidemiological models usually assume that all hosts are equally susceptible to infection. Here, we summarize models in which we relax these simplifying assumptions, thereby allowing for feedbacks between evolution, ecology, and epidemiology. One major result from these models is that, under certain general conditions, a parasite's potential for disease spread (R_0) decreases as genetic diversity for resistance increases in the host population. Moreover, R_0 can increase if we allow the parasite population to "track," or preferentially adapt to infect, common host genotypes. Feedbacks between ecology and evolution mean that as a common genotype comes to dominate the host population, the parasite population adapts to preferentially infect this genotype, increasing the prevalence of infection and the mean number of exposures to parasites per host. We further connect these findings to the major evolutionary hypothesis that coevolving parasites can favour sexual reproduction. The assumptions and conclusions of these theoretical models are supported in a natural host-parasite system, the freshwater snail *Potamopyrgus antipodarum* and its sterilising trematode parasite *Microphallus*. Field and experimental studies suggest that genetic diversity limits the spread of *Microphallus* and that host-parasite coevolution increases the prevalence of *Microphallus*. Moreover, there is evidence that *Microphallus* contributes to the maintenance of sexual reproduction in *P. antipodarum*. The ideas we review here arise from merging epidemiology and coevolutionary genetics.

Chapter 3 Wild rodents as a natural model to study within-host parasite interactions

Amy B. Pedersen & Andy Fenton

Lay summary

Individuals are typically infected by multiple parasite species. Laboratory studies show these 'coinfecting' species often interact within a host to affect the outcome of disease and treatment success. However, we have limited understanding of the occurrence or consequences of coinfection interactions in nature. Wild rodents are arguably ideal for studying coinfection, due to their abundance and tractability for repeated sampling and experiments. We review studies alongside theory, highlighting the difficulties in detecting coinfection interactions from population-level parasite data. Finally, we suggest future research that brings wild and laboratory rodent systems together, to obtain a more holistic view of the mechanisms and consequences of coinfection interactions.

Abstract

Individuals are typically coinfected with multiple parasite species - this is not only true for wildlife but also for domestic animals and humans. Laboratory studies have shown that coinfecting parasites can interact strongly within individual hosts, with potentially serious consequences for disease progression and successful treatment. However, understanding the occurrence of these interactions in natural systems, and their importance in affecting host health and parasite epidemiology in the wild, are only beginning to be understood. Rodents are arguably the ideal 'wild' model taxon to study these effects, due to their ubiquity, high abundance, amenability to capture, diagnostic assessment, and experimentation in their natural setting. Furthermore, their close phylogenetic relatedness to the standard laboratory mouse (*Mus musculus*) model, from which we have gained much of our understanding of host immune responses to infection and coinfection, mean that studies of wild rodents have great potential to advance our understanding of the dynamics and mechanisms of coinfection interactions. Here we review coinfection studies in wild rodents, and compare their findings with predictions of general coinfection theory. We show that the relationship between coinfection interactions at the within-host scale, and their pattern of association at the host population scale, can be complex, as predicted by the general theory. Hence, patterns of parasite association at the host population level can be poor predictors of the occurrence or direction of the underlying within-host interaction. We suggest that future research requires a greater understanding of the scaling relationships between the within-host mechanisms of interaction and the between-host consequences of those interactions and argue that wild rodent systems have the potential to test, inform, and advance our understanding in this important area of research into the future.

Chapter 4 **From population to individual host scale and back again: testing theories of infection and defence in the Soay sheep of St. Kilda**

Adam D. Hayward, Romain Garnier, Dylan Z. Childs, Bryan T. Grenfell, Katherine A. Watt, Jill G. Pilkington, Josephine M. Pemberton & Andrea L. Graham

Lay summary

Several thousand years ago, intrepid seafarers landed on the remote St Kilda archipelago, releasing some of their recently-domesticated sheep. The sheep remain today, unmanaged and feral, and thriving despite unreliable food supplies, adverse weather, and parasites. The interactions between the sheep and their parasites have been studied intensively since 1988. Early studies revealed the diversity of the parasite community and the impact of parasites on survival and reproduction (fitness) of the sheep. Recently, application of immunological tools has revealed the interplay between immune function, infection and fitness, giving tantalising glimpses of how natural selection shapes variation in defence against parasites.

Abstract

Why do hosts vary so much in parasite burden? How does variation in parasite burden translate to variation in host demographic rates and parasite transmission? And how, in turn, does varied transmission intensity impact selection upon immune defence of individuals? The strong theoretical foundations of disease ecology provide predictions for the answers to these questions. Yet testing such predictions with empirical data poses many challenges, since few natural host-parasite systems enable collection of sufficiently detailed data on infection, immunity and fitness at both the individual and population scales. Here, we show how the long-term ecological and genetic study of the unmanaged Soay sheep (*Ovis aries*) of St. Kilda, Scotland, has addressed fundamental questions in disease ecology, thanks to longitudinal data on parasite burden, immune defence, condition, survival, and fecundity of over 10,000 individuals. The rich individual-scale data are complemented by over 30 years of data on sheep population dynamics and genetic diversity as well as parasite dynamics and diversity. Work at the population scale has documented the range of parasite species present and demonstrated the contribution of the most prevalent and virulent parasites, gastrointestinal strongyle nematodes, to regulating sheep dynamics. Meanwhile, work at the individual scale has identified drivers of variation in parasite burden and tested hypotheses about the costs and benefits of defence in a quest to determine how natural selection has shaped immune function of the sheep. Such groundwork at each biological scale is beginning to enable the cross-scale (individual to population and back again) analysis that underpins crucial tests of disease ecology theory. We outline our progress in tackling cross-scale questions and how future research will maximise the potential of the Soay sheep system to quantify reciprocal feedbacks between parasite exposure and host defence, informing broad understanding of disease ecology.

Chapter 5 The causes and consequences of parasite interactions: African buffalo as a case study

Vanessa O. Ezenwa, Anna E. Jolles, Brianna R. Beechler, Sarah A. Budischak & Erin E. Gorsich

Lay summary

Parasites live in multi-species communities where they interact with one another in a variety of ways. However, because interactions between parasite species are often hidden, occurring at a within-host scale, the extent to which these interactions occur in nature, their relative strength and consequences are still poorly understood. This chapter reviews an emerging body of work on parasite interactions occurring in free-living African buffalo (*Syncerus caffer*). Buffalo are distributed across the African continent where they host a wide diversity of micro- and macroparasites, ranging from bacteria and viruses to helminths. Studies of three types of pairwise interactions between parasites harboured by buffalo: macroparasite-microparasite, microparasite-microparasite and macro-parasite-macroparasite reveal that interactions between parasites can have profound consequences both for individual hosts and population-level disease dynamics.

Abstract

Parasites, like their free-living counterparts, live in multi-species communities where they interact with one another in a variety of ways. Because interactions between parasite species are often hidden, unfolding at a within-host scale, the extent to which these interactions occur in nature, their relative strength and consequences are still poorly understood. This chapter reviews an emerging body of work on parasite interactions occurring in free-living African buffalo (*Syncerus caffer*). Buffalo are distributed across the African continent where they host a wide diversity of micro- and macroparasites, ranging from bacteria and viruses to helminths. Three case studies of pairwise interactions occurring between some of the most common and economically important parasites harboured by buffalo are shedding new light on the consequences of parasite interactions for individual hosts and population-level disease dynamics. First, work on interactions between macroparasites and microparasites, in this case common gastrointestinal worm infections and bovine tuberculosis (TB), suggests that immune responses of the host underlie complex interactions between these two parasites. At the individual host level, worms enhance the severity of TB infection, but at a population level this effect can limit the spread of TB. Second, an analysis of interactions between TB, a recent invader into the parasite community of buffalo, and a native microparasite, Rift Valley Fever virus (RVFV), indicates that the presence of TB makes RVFV more severe at both the individual host and population levels. Third, an investigation of how two dominant members of the worm community living within the buffalo gastrointestinal tract reassemble after perturbation reveals that the processes driving interactions between parasites can be dynamic over time. Altogether, these studies use a combination of empirical and modelling approaches to bridge the gap between individual- and population-scales and demonstrate how studies of natural populations can be used to advance our understanding of parasite interactions. Importantly, these pairwise case studies also set the stage for tackling multi-species parasite interactions which better reflect the real communities in which most parasites live.

Chapter 6 Effects of host lifespan on the evolution of age-specific resistance: a case study of anther-smut disease on wild carnations

Emily Bruns

Lay Summary

The timing of infection can have a big impact on host fitness. Becoming infected with a fatal or sterilising disease while young is much worse than becoming infected later in life, after reproducing. So, why are younger hosts generally more susceptible to disease? The first half of this chapter begins by examining the case of anther-smut, a sterilising fungal disease of wild carnations. New seedlings are significantly more susceptible to disease than older adults. While the plant has the capacity to evolve higher levels of resistance, the majority of families are susceptible. The second half of the chapter develops a mathematical model to understand the costs, benefits, and life history traits driving the evolution of age-specific resistance.

Abstract

A large class of human and wildlife diseases are dependent on juvenile hosts for their transmission because younger hosts are typically more susceptible to disease. While numerous studies have investigated the epidemiological consequences of juvenile susceptibility, the question of why species retain such high susceptibility in the juvenile stage remains an evolutionary puzzle. Indeed, life history theory predicts that hosts should evolve to be more resistant as juveniles than as adults since early infection is costlier to the host. Studies of anther-smut, (a pollinator-vectored, sterilising disease) on wild carnations show that disease persistence in nature is strongly dependent on the presence of a highly susceptible juvenile class. However, while there is evidence of genetic variation in juvenile resistance, the majority of plant families are highly susceptible at this stage, indicating that juvenile resistance may be less beneficial than previously assumed. To understand how the costs and benefits of resistance and life history traits affect the evolution of age specific resistance I developed a general analytical model of age-specific resistance. The model shows that if there is genetic variation for the onset of resistance, selection and numerical feedbacks will often drive the evolution of adult resistance but maintain susceptibility at the juvenile stage. I discuss the implications of these results for understanding disease dynamics and the evolution of pathogen transmission modes.

Chapter 7 **Sexually transmitted infections in natural populations: what have we learnt from beetles and beyond?**

Ben Ashby, Jordan E. Jones, Robert J. Knell & Gregory D.D. Hurst

Lay Summary

Sexually transmitted infections (STIs) are common in animals. Because they are transmitted during host mating, the epidemiology of these infections is expected to depend on their animal host's mating biology – the mean and variance in the number of sexual partners, the patterns of association between individuals – and to be less dependent than other diseases on their host's population size. STIs are also predicted to impact on the evolution of mating behaviour and mating systems. We examine the extent to which studies of wildlife STIs – in particular ladybird-sexually transmitted mite interactions – support or refute these predictions. We see that high mating rates promote STI spread and that the pattern of mating contacts is likely important for preventing STIs from causing extinction. Initial models of STI spread in which host mating rate is insensitive to population size are now being replaced with more realistic formulations where mating rate declines at low population size. However, there is surprisingly little evidence for the STI causing the evolution of either mate choice or a reduction in mating rate.

Abstract

Sexually transmitted infections (STIs) can be found in a wide range of invertebrate and vertebrate hosts. Theory makes extensive predictions with respect to the dynamics and evolutionary ecology of these infections. Their epidemiology is predicted to vary with the mean and variance in the number of mating partners, and in more refined models, contact and social structure. Weak dependence of mating rate on host density leads to the prediction of density-independent dynamics, including the possibility that sterilizing infections could drive their hosts extinct. The impact of infection on the host is predicted to select for mate choice against infected partners, and for reduced mating rates. We examine these predictions against STIs in nature, with a particular focus on studies of beetle-ectoparasitic mite interactions. The *Adalia bipunctata* (two spot ladybird) – *Coccipolipus* (mite) interaction has given particularly rich insights, with the ease of scoring infection and mating activity in natural populations enabling detailed documentation of dynamics. Laboratory study has further allowed precise estimation of transmission parameters to inform models, and allowed focused analysis of behaviour. The use of replicated population microcosms permits controlled emulation of natural systems. These studies have confirmed the core impact of mating rate on STI dynamics, but revealed unexpected drivers such as food supply (positively driving mating rate) and sex ratio (enhancing spread and producing male-biased prevalence), alongside constraints on spread from host phenology. They have challenged the independence of STI dynamics with respect to host density, but have largely failed to support predictions that STIs drive the evolution of mate choice and mating rate.

Chapter 8 **Using insect baculoviruses to understand how population structure affects disease spread**

Bret D. Elderd & Greg Dwyer

Lay Summary:

An understanding of the drivers of epidemics in animal populations and the long-term dynamics of animal host-pathogen interactions is often best achieved by using field data to test mechanistic models of disease spread. Many host-pathogen interactions can only be studied using observational data or laboratory experiments, but in our work we have shown that insect baculoviruses can be used in field transmission experiments that allow for an understanding of how disease transmission is affected by a range of abiotic and biotic factors. The models show and the data confirm that transmission dynamics and host-pathogen evolution are often governed by population structure, such as heterogeneity in infection risk, or changes in population structure, such as pathogen polymorphism, either of which may in turn depend on host genetic variation or host resource quality. Emerging areas of baculovirus research include coevolutionary interactions between hosts and pathogens and the development of statistical techniques that permit inferences based on a combination of ecological and evolutionary data.

Abstract:

Current approaches to understanding infectious disease dynamics often emphasize studies of multiple hosts or multiple pathogens, but for many diseases, population structure may play a more important role. Changes in population structure may stem from heterogeneity of infection risk in the host population, pathogen polymorphism, spatial structure, or size structure in the host population. Here we review our previous work demonstrating the effects of population structure on baculoviruses of two Lepidopteran insects in North America, the invasive forest-defoliating gypsy moth (*Lymantria dispar*) and the crop pest, the fall armyworm (*Spodoptera frugiperda*). We focus on heterogeneity in infection risk, such that some hosts are at higher risk than others, which has consequences for both single epizootics and long term host-pathogen population cycles. The simple biology of baculoviruses, and the small size of their insect hosts, means that insect-baculovirus interactions provide experimentally tractable systems for testing mechanistic models. Our research has therefore combined mechanistic models with data by using a combination of statistical model selection, Bayesian statistics, and time-series probes. This approach has allowed us to show how host variation affects disease dynamics and pathogen coexistence, and how variation is in turn affected by host-plant resource quality, climate change, and the size structure of the host population. Future avenues of research include a consideration of host-pathogen coevolution, and how such coevolution may be modulated by environmental factors that vary across the landscape. We also discuss how Bayesian mixture models can make it possible to combine multiple sources of data collected across a range of spatial and temporal scales. Our work illustrates the usefulness of an iterative process of comparing models to data that allows for a detailed understanding of transmission processes.

Chapter 9 **Infection and invasion: study cases from aquatic communities**

Melanie J. Hatcher, Jamie T.A. Dick, Jamie Bojka, Grant D. Stentiford, Paul Stebbing & Alison M. Dunn

Lay summary

Invasive species drive biodiversity loss and lead to changes in parasite-host associations. We review empirical and theoretical research into parasites in biological invasions, using our aquatic crustacean study systems. We focus on the effects of parasitic infection on host traits (behaviour and life history). The trait effects of parasites can mediate a range of host trophic interactions including predation, intraguild predation and cannibalism, and hence can mediate invasion success and impacts. We highlight challenges of managing invasive parasites and argue that policy should take into account the strong links between biological invasions, wildlife diseases and threats from alien parasites.

Abstract

Invasive species drive biodiversity loss and lead to changes in parasite-host associations. Parasites in turn are linked to invasions; they can mediate invasion success and outcomes for the recipient community. In this chapter, we review our theoretical and empirical research into parasites in biological invasions, focusing on our freshwater invertebrate study system. We focus on the effects of parasitic infection on host traits (behaviour and life history) which can mediate a range of native/invader trophic interactions. We review our evidence from the field and laboratory of parasite-driven changes in predation, intraguild predation and cannibalism. Our theoretical work reveals that the trait- mediated effects of parasites can be as powerful as classical density effects and their impact on the host's trophic interactions merits deeper consideration. We also report on tantalising evidence of broader cascading effects that warrant deeper study. The process of biological invasion can lead to altered parasite-host associations. Focusing on amphipod invasions, we find patterns of parasite introduction and loss that mirror host invasion pathways, but also highlight the risks of introduction of invasive parasites. Horizon scanning and impact predictions are vital in identifying future disease risks, potential pathways of introduction and suitable management measures for mitigation. We highlight challenges of predicting and managing invasive parasites and argue that policy should take into account the strong links between biological invasions, wildlife diseases and threats from alien parasites.

Chapter 10 Parasite mediated selection in red grouse – consequences for population dynamics and mate choice

Jesús Martínez-Padilla, Marius Wenzel, François Mougeot, *Lorenzo Pérez-Rodríguez, Stuart Piertney & Stephen M. Redpath*

Lay summary

Parasites inflict considerable costs on their hosts, reducing condition and fitness components such as life-span, fertility and survival. These individual level effects can also have population level consequences, influencing recruitment, population size and growth rate and overall viability. Over decades, we have studied how the Scottish red grouse (*Lagopus lagopus scotica*) is affected by its main intestinal threadworm parasite (*Trichostrongylus tenuis*) at both the individual and population levels. Here we present a synthesis of our current understanding, emphasizing how parasites are central to shaping both the ecology and genetics of natural populations.

Abstract

Parasites inflict considerable, multifaceted costs on their hosts, the consequences of which are manifest at the individual and population levels and over ecological and evolutionary timescales. Any capacity to fully understand the eco-evolutionary dynamics of the host-parasite interrelationship requires first an appreciation of how parasites affect individual fitness, survival and reproductive potential; then how these combine to influence population demography, dynamics and viability; then finally how these individual and population level processes drive microevolutionary processes that define both natural and sexual selection. Here we synthesize the large body of work we and others have amassed on the interrelationship between the red grouse (*Lagopus lagopus scotica*) and its main parasite, the gastrointestinal nematode parasite *Trichostrongylus tenuis*. At the individual level, we emphasize how parasites impose a physiological cost, measured through immunosuppression and increased oxidative stress, and also how the impact of these costs on the host vary depending on environmental and social contexts. We describe how parasite infection constrains the expression of sexually selected traits, such as calling rate and size and colouration of combs for both males and females. We then summarise how interrelationships between the parasite, its host and the environment shape host population demography and dynamics, focussing on social structure within populations and cyclic population dynamics. Finally, we show how genetic analyses in red grouse suggest that nematode burden is moderately heritable (c. 29%) and underpinned by a potentially large array of genes involved in the immune system, energy balance and broader homeostatic processes. However, there is no clear association between allele frequencies among populations and differences in nematode burdens. Thus, it is possible that any beneficial alleles for parasite resistance cannot spread through the population because of the strong diversifying effects of gene flow and genetic drift.

Chapter 11 Emergence, transmission and evolution of an uncommon enemy: Tasmanian devil facial tumour disease

Menna E. Jones, Rodrigo Hamede, Andrew Storfer, Paul Hohenlohe, Elizabeth P. Murchison & Hamish McCallum

Lay summary

Devil facial tumour disease (DFTD) emerged about 20 years ago in Tasmanian devils, the world's largest marsupial carnivore that is endemic to the island of Tasmania. DFTD is a transmissible cancer that is spread through injurious biting, mostly during the mating season. The Tasmanian devil—DFTD host—pathogen system provides a rare opportunity to study a wildlife disease in all stages of existence across the entire geographic range of a natural host species (from pre-emergence to emergence, to post-emergence decline, and potentially even endemism and host recovery), and the evolutionary responses of both host and tumour.

Abstract

In just over 20 years since its emergence, devil facial tumour disease (DFTD) has spread throughout most of the geographic range of the Tasmanian devil (*Sarcophilus harrisii*), causing >90% local population decline, an overall decline of >80% and cascading effects on the ecosystem. A decision tree was developed to guide research and management of this novel transmissible cancer, in which live tumour cells are the aetiological agent. The Tasmanian devil—DFTD host—pathogen system provides a rare opportunity to study a wildlife disease in all stages of existence across the entire geographic range of a natural host species (from pre-emergence to emergence, to post-emergence decline, and potentially even endemism and host recovery).

Despite predictions of extinction from simple deterministic models, and sustained >90% decline and 50% disease prevalence, devils persist in the wild. State-space models show that individuals with higher fitness, the larger more dominant individuals responsible for most of the biting, are more likely to become infected themselves. Individual-based models, in which demographic parameters depend on the size of the tumours carried by individual hosts, reveal that DFTD epidemics operate on a much slower timescale than those of viral or bacterial wildlife diseases. Following an initial epidemic peak, the consequences for a general epidemic may be coexistence, even in the absence of evolutionary changes in either host or pathogen.

Multiple lines of evidence show that Tasmanian devils are evolving in response to DFTD. Rapid evolution is occurring in as little as four generations in candidate genes associated with cancer and immune function, suggesting evolution of resistance. Tumour regressions and confirmed antibody production are now documented, and force of infection is reducing. Conservation efforts are now shifting from managing for extinction to managing for persistence.

Chapter 12 Bovine tuberculosis in badgers: sociality, infection and demography in a social mammal

Jenni L. McDonald, Richard J. Delahay & Robbie A. McDonald

Lay summary

As an important reservoir of bovine tuberculosis, badgers have been the subject of much field research. We describe how the Woodchester Park longitudinal study of the ecology and epidemiology of tuberculosis in badgers has provided insight into three key concepts. First, the importance of distinguishing between infected and infectious individuals, providing an understanding of the variability in infection states beyond traditional classifications. Second, pairing epidemiological data with tracking techniques to explore how spatial structuring and host-host interactions influence the spread of disease across a population. Finally, using modelling approaches to explore demographic mechanisms underpinning the persistence of infected badger populations.

Abstract

Badgers are an important reservoir of bovine tuberculosis in the UK. Using research from the Woodchester Park longitudinal capture-mark-recapture study of badgers and bovine tuberculosis, we discuss how the combination of individual epidemiological data, tracking studies and modelling frameworks has enabled the exploration of host-pathogen theories in relation to badger life history and disease ecology. This chapter explores three key concepts. First, we focus on heterogeneity in host susceptibility and infectiousness, which are two components of the superspreader phenotype. Badgers can experience a variety of disease states, each with differing levels of infectiousness and mortality rates. Studies pairing empirical data with modelling approaches suggest sex differences are underpinned by immunological mechanisms. Second, we discuss how studies have moved away from the simplistic assumption of randomly mixing homogeneous populations, and towards recognition of heterogeneity in host association patterns at a group-, class- and individual level. Interactions between individuals are non-random with high within-group contacts and lower between-group contact rates. Contact heterogeneity amongst individuals is fundamental to understanding what drives (or restricts) the spatial spread of disease through a population. Finally, we explore a demographic perspective on disease ecology, revealing how demographic intricacies including compensatory recruitment rates provide further understanding of the mechanisms underpinning the persistence of infected badger populations. The understanding gained from longitudinal studies of host-pathogen field systems plays an important role in the development of ecological and epidemiological theory and usefully informs the development of evidence-based disease control strategies.

Chapter 13 ***Mycoplasma ovipneumoniae*** **in bighorn sheep: from exploration to action**

Kezia Manlove, Emily S. Almberg, Pauline L. Kamath, Raina K. Plowright, Thomas E. Besser & Peter J. Hudson

Lay summary

Bighorn sheep in the western United States suffer from an infectious pneumonia that constrains their populations and challenges managers. A major challenge in this system is that diagnosis was historically based on a syndrome, as opposed to a particular known agent. Recent work has uncovered a primary driving agent, *Mycoplasma ovipneumoniae*, and this new knowledge has substantially increased research and management opportunities for this system. Here, we present our current understanding of the in-host and between-host processes driving bighorn sheep pneumonia, and describe how this new information has changed how we research and manage this system.

Abstract

Some of the greatest successes in infectious disease control rest on empirically grounded models of human and livestock infections. In contrast, disease control in wildlife has not always been as successful. In particular, the timely translation of knowledge into proposed management actions still remains a challenge in several important wildlife disease systems, one of which is pneumonia management in bighorn sheep throughout the North American West. Although pneumonia was recognized as a major impediment to bighorn sheep conservation more than 80 years ago, a series of challenges stymied the management decision-making process including disagreements about the primary causal agent, questions over the immunological response of adapted and novel hosts, and uncertainties in host spatial ecology. Despite these past obstacles, recent advances from long-term, intensive studies of marked individual bighorn sheep have motivated new interest in research-driven strategies for disease management in the bighorn system. This system provides an unusual opportunity to study an emerging pathogen disproportionately impacting immature animals through infections that originate from asymptomatically infected adult hosts. In this chapter, we tell the story of bighorn sheep pneumonia, emphasizing the obstacles that historically hindered decision making, the biological or logistical constraints underlying each decision point, and the particular empirical insights that clarified each constraint.

Chapter 14 Manipulating parasites in an Arctic herbivore: gastrointestinal nematodes and the population regulation of Svalbard reindeer

R. Justin Irvine, Steve D. Albon, Audun Stien, Odd Halvorsen & Anja M. Carlsson

Lay summary

Parasite regulation of their host numbers has been suggested in theory but rarely demonstrated. Our multi-year study of Svalbard reindeer measured differences in both the abundance of their two main parasite species and the effect of treating some reindeer with anti-parasite drugs on fecundity and survival. Only the abundance of the parasite *Ostertagia gruehneri*, transmitted over the summer depressed host fecundity and models show that this is sufficient to regulate the reindeer population. Surprisingly, the other main parasite *Marshallagia marshalli* was transmitted during the harsh arctic winter, but had little impact on fitness. Our results highlight the importance of long-term monitoring and field experiments for understanding host-parasite interactions.

Abstract

Living in an environment with no functional predators or competitors, the main drivers of Svalbard reindeer population dynamics are likely to be limited food resources, periods of harsh winter weather and their abundant parasitic nematode infections. Teasing out the demographic impact of these macroparasites on their hosts requires integrating three different approaches: i) field observation to document the life history and abundances of both parasite and hosts, ii) experimental manipulation of infection to quantify the effect of parasite intensity on host fitness, iii) appropriate population models of density-dependent transmission. Thus, over many years, we monitored the reindeer population and the intensity of parasites in culled reindeer. Also, in April, we treated a randomly selected group of reindeer with an anthelmintic and compared their fitness with a control group. The two main abomassal nematode species differed substantially in life-histories. *Ostertagia gruehneri* infected the reindeer over the summer, as expected. In contrast, *Marshallagia marshalli* transmission was limited to the harsh arctic winter. These life-history differences imply that our experimental treatment only affected *O. gruehneri* and showed that reindeer fecundity depended on the intensity of infection with *O. gruehneri*, which varied between years and was positively related to host population size. Our modelling of this interaction suggested a role for *O. gruehneri* in reindeer population regulation. Subsequent experiments, with a delayed-anthelmintic treatment, designed to manipulate *M. marshalli* abundances over the winter, provided little evidence of an impact of this parasite on host population dynamics. Our study demonstrates the importance of understanding parasite life-histories and the value of long-term experimental manipulations in order to test ecological theory on the role of parasites in the population regulation of wild host – particularly when dealing with mixed natural infections, as is common in both domestic and wild host parasite systems.

Chapter 15 The ecological and evolutionary trajectory of oak powdery mildew in Europe

Marie-Laure Desprez-Loustau, Frédéric M. Hamelin &Benoit Marçais

Lay summary

Powdery mildew is a relatively new yet already common foliar disease of European oak trees, caused by a complex of fungal species that coevolved with Asian oaks and were introduced to Europe last century. To explore its eco-evolutionary dynamics, we developed a semi-discrete model combining a SIR model in the epidemic phase and a discrete-time model between years. This model, based on a within year - between year transmission trade-off, adequately described two main features of the disease: seasonality and the occurrence of a pathogen complex. We discuss refinement of this model and other modelling approaches for the evolution of virulence and resistance in a context of changing environment.

Abstract

Oak powdery mildew in Europe is an example of disease in a wild perennial plant which has displayed dramatic changes over the course of a century; from typical invasion dynamics after pathogen introduction into a new area, characterized by severe damage, to a new equilibrium with moderate damage. Several non-mutually exclusive hypotheses could account for this, including pathogen evolution towards lower virulence, a reciprocal increase in oak population resistance, and both environmental biotic (phyllosphere microbes) and abiotic (climate) factors. We show that understanding the pathosystem requires the accounting of both seasonality (i.e. succession of epidemic and inter-epidemic phases linked to availability of susceptible leaves) and the occurrence of a pathogen complex with several cryptic fungal species differing in their life history traits. Observational data suggests that the severity of annual epidemics is linked to inter-annual pathogen transmission, including winter survival and the infection success of the primary inoculum in spring. Climate-driven phenological synchrony between host and pathogen in spring thus appears to be a critical factor. Several cryptic Erysiphe species are associated with the disease and co-occur at multiple spatial scales from individual leaves to continent. A semi-discrete model combining a SIR model in the epidemic phase and a discrete-time model between years, based on a within season (intra-epidemic) - between season (inter-epidemic) transmission trade-off, adequately describes seasonality and the coexistence of pathogen species. We discuss the refinement of this model, through the introduction of age classes in the host population in particular, and other modelling approaches for the evolution of pathogen virulence and host resistance in a context of changing environment.

Chapter 16 Healthy herds or predator spreaders? Insights from the plankton into how predators suppress and spread disease

Meghan A. Duffy, Carla E. Cáceres & Spencer R. Hall

Lay summary

Sometimes predators suppress disease in their hosts, but other times they spread disease. Here, we explore the factors that drive these divergent outcomes, laying out a framework explaining different mechanisms by which predators can influence disease in their prey. We review evidence for these different mechanisms from a variety of predator-prey/host-parasite systems, but focus particularly on the prey/host species that has been the focus of our work for the past 15 years: the ecologically important lake crustacean, *Daphnia*. In this system, bluegill sunfish serve as a "healthy herds" predator, reducing disease in our focal host, *Daphnia dentifera*. Phantom midge larvae, on the other hand, are "predator spreaders", fueling disease outbreaks in *Daphnia*. A key question that continues to motivate our research is: what determines whether predators promote or prevent disease?

Abstract

Predators are often thought to decrease the size of disease outbreaks, particularly through selective predation on infected hosts and/or predation on free-living infectious stages of parasites. But we also see cases in nature where higher predator densities are associated with more disease, not less. How and why do predators sometimes fuel disease outbreaks but other times thwart them? Answering this question could help explain spatial and temporal variation in disease, and could also explain why attempts to control disease by manipulating predators sometimes fail. Here, we lay out eight mechanisms by which predators can suppress or spread disease in prey populations. We explore each of these mechanisms generally and also review evidence from the study system that has been the focus of much of our research. This system focuses on the crustacean *Daphnia dentifera*, a dominant herbivore in lake food webs in the Midwestern United States. *D. dentifera* is prey to bluegill sunfish and phantom midge larvae, as well as host to a virulent fungal pathogen. We review evidence for bluegill sunfish as "healthy herds" predators that reduce disease, and for midge larvae as "predator spreaders" that fuel disease outbreaks. We find that both predators can impact disease via multiple mechanisms. Bluegill feed selectively on infected hosts, and also depress disease in *Daphnia* by reducing the density of midge larvae which spread disease. Bluegill also increase the abundance of *Ceriodaphnia* which reduce disease. Midge larvae increase disease in their hosts, in part by releasing spores into the water column where they can be consumed by additional hosts. We call for further research aimed at uncovering the relative importance of the different mechanisms, as well as into how global change might alter the impacts of these predators on disease. Such studies should allow us to better predict how and when predators should suppress or spread disease.

Chapter 17 Multi-trophic interactions and migration behaviour determine the ecology and evolution of parasite infection in monarch butterflies

Jacobus C. de Roode, Sonia Altizer & Mark D. Hunter

Lay summary

Monarch butterfly caterpillars are specialist feeders on milkweeds, from which they derive chemicals that make them toxic to their predators, and monarchs advertise this toxicity with their bright colouration. When temperatures fall and day length shortens, eastern North American monarchs embark on an autumn journey that spans thousands of kilometres to colonize their overwintering sites in Mexico. Studies over the last two decades have shown that the sequestration of toxic chemicals and autumn migration have important impacts on infectious disease in monarch butterflies, with toxic milkweeds reducing parasite infection and strenuous migration weeding out the most heavily infected individuals.

Abstract

The monarch butterflies (*Danaus plexippus*) is most famous for its warning colouration and seasonal migration. Monarch caterpillars are specialist feeders on milkweeds, from which they sequester toxic chemicals called cardenolides. Cardenolides protect monarchs against vertebrate predators and their toxicity is advertised through bright colours. North American monarchs undergo an annual migration, during which millions of butterflies traverse thousands of kilometres from the United States and Canada to overwinter in central Mexico. The last two decades have shown that the iconic biological concepts of sequestration and migration combine to make monarchs an ideal system with which to study the effects of multi-trophic interactions and migration behaviour on the ecology and evolution of infectious disease. Monarchs are commonly infected with a virulent protozoan parasite, and our studies have shown that milkweed chemicals reduce parasite growth, transmission, and virulence. Consequently, monarchs can use toxic milkweeds as a form of medicine to reduce infection in their offspring. While medication behaviour reduces parasite growth and disease in individual monarchs, the use of medicinal milkweeds may also select for more virulent parasites. Our studies have also shown that seasonal migration is an important determinant of parasite prevalence, through two mechanisms: migratory culling is the process by which the most heavily infected individuals are weeded out during the autumn migration, while migratory escape is the phenomenon by which monarchs escape contaminated environments, and thereby reduce the probability of infection. Recently, well-intentioned but ill-advised conservation efforts have increased the planting of non-native medicinal milkweeds in North America, while monarchs have increasingly formed sedentary populations. This has reduced rates of migration. These factors have already increased parasite prevalence in non-migratory populations. Integrating studies on multi-trophic interactions and migration behaviour are now necessary to determine their long-term effects on parasite dynamics and host and parasite evolution in natural monarch populations. Ultimately, this integration will provide insights into the general importance of multi-trophic interactions and migration for disease ecology and evolution.

Chapter 18 When chytrid fungus invades: integrating theory and data to understand disease-induced amphibian declines

Mark Q. Wilber, Pieter T.J. Johnson & Cheryl J. Briggs

Lay summary

The emerging amphibian fungal pathogen *Batrachochytrium dendrobatidis* (Bd) has led to severe amphibian declines around the globe. One of the challenges when attempting to mitigate the effects of Bd on amphibian populations is that different populations can show drastically divergent outcomes following Bd invasion. These include an increase in amphibian population density, no discernible change in population density, a decrease in density, and even population-level extinction. Here we integrate extensive data from amphibian-Bd systems and epidemiological theory to build a framework for predicting when and why amphibian populations show different population-level trajectories upon Bd invasion, ultimately allowing us to better understand and manage amphibian declines and recoveries.

Abstract

Amphibian populations around the globe are experiencing declines, many of which are driven by the fungal pathogen *Batrachochytrium dendrobatidis* (Bd). However, different species of amphibians, as well as divergent populations of the same species of amphibian, can show drastically different responses to Bd invasion. In this chapter we answer three questions: 1) What are the potential trajectories of amphibian host populations following Bd invasion? 2) How are each of these trajectories influenced by the transmission dynamics and load dynamics governing an amphibian-Bd system? 3) How do ecological, evolutionary, and environmental factors affect both Bd transmission and Bd load dynamics, which in turn influence the population-level outcome of amphibian hosts? We build a general framework that identifies eight population-level trajectories that amphibian populations can take upon Bd invasion that are a result of five different branch points. Each of these branch points is affected by either the transmission dynamics or the load dynamics underlying the system. By integrating relevant disease ecology theory and empirical data, this framework can be used to guide context-dependent management strategies for amphibian populations infected with Bd. While this framework is motivated by amphibian-Bd systems, we anticipate that it will also provide a useful lens through which to view the relative importance of transmission and load dynamics in other host-pathogen systems.

Chapter 19 Ecology of a marine ectoparasite in farmed and wild salmon

Stephanie J. Peacock, Andrew W. Bateman, Brendan Connors, Sean Godwin, Mark A. Lewis & Martin Krkošek

Lay summary

The environment on farms can be very different from nature, and these differences can influence the ecology and evolution of hosts and parasites. When farmed and wild hosts share parasites, understanding and predicting the outcomes of host-parasite interactions can be difficult due to their complexity. This chapter examines the case study of sea-louse transmission between farmed and wild salmon, which has implications for wild salmon ecology and conservation, and the traits of the parasites themselves.

Abstract

The ecology and evolution of parasites can differ greatly between wild and domestic host environments. Parasitism can affect every aspect of wildlife ecology, from predator avoidance and competition for food, to migrations and reproduction. In the wild, these ecological effects of parasites can have implications for both host fitness and parasite dynamics. In contrast, domestic environments are typically characterized by high host densities, low host diversity, and veterinary interventions, and are not subject to processes like predation, competition, and migration. When wild and domesticated hosts interact via shared parasite populations, understanding and predicting the outcomes of parasite ecology and evolution for wildlife conservation and sustainable farming can be a challenge. This chapter describes the ecology and evolution of ectoparasitic copepods – sea lice – that are shared by farmed and wild salmon, and the insights that experiments, fieldwork, and mathematical modeling have generated for theory and applied problems of host-parasite interactions over the course of a long-term study in Pacific Canada. The salmon and sea lice host-parasite system provides a rich case study for examining the ecological context of host-parasite interactions and for shedding light on some the principal challenges of parasite management for wildlife health and conservation.

Chapter 20 Mycoplasmal conjunctivitis in house finches: the study of an emerging disease

André A. Dhont, Andrew P. Dobson & Wesley M. Hochachka

Lay summary

A single successful host jump of the bacterium *Mycoplasma gallisepticum* from domestic poultry to free-living house finches (*Haemorhous mexicanus*) caused a major epidemic in its new host resulting in a decline of house finch abundance by half in eastern North America within three years of the disease becoming established. Once established, mycoplasmal conjunctivitis showed regular seasonal fluctuations in prevalence driven by the interaction between house finch breeding, seasonal changes in flocking behaviour, and partial immunity of hosts after recovery. As it increased in abundance and spread geographically *M. gallisepticum* evolved rapidly in virulence illustrating an unexpected pattern of increasing in virulence where endemic, and decreasing in virulence at the expansion front. This provides an interesting study system to understand selection pressures on virulence and patterns of evolution in an emerging pathogen.

Abstract

Mycoplasma gallisepticum is a bacterium that causes disease in poultry worldwide. Around 1994, it emerged as a novel pathogen in the house finch (*Haemorhous mexicanus*) resulting in an epidemic of mycoplasmal conjunctivitis across North America. This was the result of a single successful host jump from poultry to house finches. With the help of thousands of Citizen Scientists who participated in the House Finch Disease Survey it was possible to document how the epidemic spread rapidly across eastern N. America, causing a monotonic decline in host abundance by about half. Once established disease prevalence showed regular seasonal variation with a late summer/early autumn peak, a mid-December minimum, followed by a late winter peak and a breeding season minimum. These observed seasonal bimodal peaks in disease prevalence require seasonal reproduction (providing an autumn pulse of naïve hosts) and winter social aggregation as well as partial immunity of recovered birds. *M. gallisepticum* virulence evolved rapidly: virulence in eastern populations increased once the disease had become endemic. The strain that successfully established in western North America was of much lower virulence, but once established there it again increased in virulence. Our data and models show that depending on the selection pressures encountered virulence may evolve in opposite directions. A detailed three-year capture-mark-release-recapture (CMRR) study of colour-ringed birds showed that disease decreased survival and mobility and that a high proportion of birds recovered; we also observed that re-observation rates of clinically diseased birds are often different from those of asymptomatic birds. For that reason, only calculations in which this effect is included allow an accurate estimate of disease prevalence in a population. A detailed understanding of the ecology and genetics of an emerging disease at multiple scales are required to predict how emerging diseases will impact their host.

Chapter 21 **Heterogeneities in infection and transmission in a parasite-rabbit system: key issues for understanding disease dynamics and persistence**

Isabella M. Cattadori, Ashutosh Pathak & Brian Boag

Lay summary

There is often large variation in the level of infection among individual hosts. While this can be caused by the way hosts react to parasites, some of this variation can also be generated by external disturbance to both parties. We examined the effect of common sources of disturbance, such as climate changes, co-infection and anthelminthic treatment, on the dynamics of infection and shedding in a parasite-rabbit system, using a combination of field and laboratory studies with both analytical and modelling approaches. Findings suggest that different disturbances alter different components of the host-parasite interactions and generate complex patterns of infection at diverse temporal scales. These perturbations can ultimately alter the risk of infection and parasite persistence over time and space.

Abstract

The severity and persistence of an infection in a host population is strongly affected by the variation in susceptibility among hosts. External disturbance can exacerbate or reduce individual variation by affecting the interactions between the host and its parasites and, in so doing, the dynamics of infection and transmission. We investigated the impact of three sources of disturbance: climate changes, the presence of a second parasite species and anthelmintic treatment, on the dynamics of infection and shedding of three common parasites of the European rabbit (*Oryctolagus cuniculus*). Data were collected from long-term field studies and laboratory experiments and analysed using mathematical modelling and analytical tools. Our studies indicate that all these types of perturbation affect host-parasite interactions by altering the intensity of infection and/or the degree of parasite shedding in a non-linear way. They also generate patterns of infections that could not have been predicted in the absence of these disturbances or from performing analyses at a different temporal scale. Modelling simulations confirmed the complexity of the processes involved and identified the critical interactions shaping the patterns observed. Given the ecological and evolutionary relevance of these disturbances, more attention should be given to their long-term impact on parasite persistence and disease spread.

Chapter 22 Sylvatic plague in Central Asia: a case study of abundance thresholds

Mike Begon, Stephen Davis, Anne Laudisoit & Jonas Reijniers

Lay summary

For an infection to exist within a population there must be sufficient hosts. We refer to this as the abundance threshold of the infection. This, though, is an idea that has arisen largely from theoretical considerations. Actual examples are rare, especially for wildlife diseases. One of the best comes from plague in central Asia, where it is spread between great gerbils by fleas, and may sometimes be passed to humans (bubonic plague). Detailed study of this system calls into question the simplest (theoretical) views of abundance thresholds. The realities are more complex – and more interesting.

Abstract

Abundance thresholds – the abundance of hosts that must be exceeded for an infection to invade and persist within a population – are of fundamental importance in our attempts to understand the dynamics of infection in wildlife. Identifying and manipulating these thresholds may also have substantial applied significance. The plague system in the Pre-Balkhash region of Kazakhstan (part of a larger plague focus in central Asia) has been subject to particularly extensive study, including an unusually thorough investigation of the nature and importance of an abundance threshold for the infection. Great gerbils (*Rhombomys opimus*) are the main reservoir host, with plague transmitted between them by a variety of flea species. Initial work was able to identify such a threshold from time series data, with great gerbil abundance being measured by their level of occupancy (the proportion of the burrow systems in the landscape supporting an extended family group). However, this and subsequent refinements of the threshold were better at predicting the absence of plague (below the threshold) than in guaranteeing its presence (above). Further analysis indicated that the threshold was a critical point in the percolation of plague across the landscape, rather than in a mass-action random mixing process, as conventionally assumed. The performance of the threshold was also improved by incorporating both gerbil and flea abundance to generate a hyperbolic threshold curve. More generally, detailed study of a natural system such as this calls into question the simplest of views of what an abundance threshold might be, generated by standard epidemiological models. The realities are that invasion and persistence should be distinguished; that abundances may need to be integrated over time and combined for all agents in a system; and that there is more than one way of generating a threshold but all have their roots in a sufficiency of contacts.